UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/991,799	11/23/2001	George Jackowski	2132.086	5599

21917 7590 12/21/2007

MCHALE & SLAVIN, P.A.
2855 PGA BLVD
PALM BEACH GARDENS, FL 33410

EXAMINER	
CHERNYSHEV, OLGA N	

ART UNIT	PAPER NUMBER
1649	

MAIL DATE	DELIVERY MODE
12/21/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE BOARD OF PATENT APPEALS
AND INTERFERENCES

Ex parte GEORGE JACKOWSKI and JOHN MARSHALL

Appeal 2007-3905
Application 09/991,799
Technology Center 1600

Decided: December 21, 2007

Before DEMETRA J. MILLS, LORA M. GREEN, and NANCY J. LINCK, *Administrative Patent Judges*.

GREEN, *Administrative Patent Judge*.

DECISION ON APPEAL

This is a decision on appeal[1] under 35 U.S.C. § 134 from the Examiner's final rejection of claim 1. We have jurisdiction under 35 U.S.C. § 6(b). The claim reads as follows:

---

[1] This Appeal is related to Appeal Nos. 2007-3735, USSN 09/993,344, and 2007-3904, USSN 09/991,796, which are decided concurrently with this Appeal.

1. An isolated biopolymer marker consisting of amino acid residues 2-18 of SEQ ID NO: 1 which evidences a link to Alzheimer's disease.

We reverse the rejections of record, but raise other issues that the Examiner should consider upon return of the administrative file.

BACKGROUND

According to the Specification:

> This invention relates to the field of characterizing the existence of a disease state; particularly to the utilization of mass spectrometry to elucidate particular biopolymer markers indicative or predictive of a particular disease state, and most particularly to specific biopolymer markers whose up-regulation, down-regulation, or relative presence in disease vs. normal states has been determined to be useful in disease state assessment and therapeutic target recognition, development and validation.

(Specification 1.)

Samples are collected from an individual, at one point in time or at different points in time (Specification 31), and are resolved using polyacrylamide gel electrophoresis (*id.* at 38). The protein bands are cut from the gel, and are cleaved into fragments using proteolytic enzymes (*id.*). The peptides are collected and purified by reversed phase chromatography, and then subject to identification by mass spectrometry (Specification 38-39).

The Specification teaches further:

> The human genome contains the genes that encode all proteins. The proteolytic cut sites within all these proteins can be predicted from the translated amino acid sequence. The mass of the peptides that result from the predicting cut sites can be calculated. Similarly, the fragmentation pattern from each hypothetical peptide can be predicted. Thus, we can

> conceptually digest the proteins within the human proteome and fragment them.
>
> When a peptide has been "sequenced" it is understood that the peptide fragment has been purified by one of the methods above, i.e. Time of flight (TOF) or by chromatography, before fragmenting it with gas to produce the peptide fragments. The original peptide mass and fragmentation pattern obtained is then fit to those from the theoretical digestion and fragmentation of the genome. The peptide that best matches the theoretical peptides and fragments and is biologically possible, i.e. a potential human blood-borne protein, is thus identified. It is possible to identify plural targets in this fashion.

(Specification 39-40.)

As to the peptide of SEQ ID NO:1, the Specification teaches:

> As a result of these procedures, the disease specific markers plasma protease (C 1) inhibitor having a molecular weight of about 1826 daltons and a sequence of SEQ ID NO: 1, molecular weight of about 1560 daltons and a sequence of SEQ ID NO: 2, molecular weight of about 1186 and a sequence of SEQ ID NO: 3 (see Fig. 1, Band 2) related to [sic. Alzheimer's] Alzheimers disease were found.

(Specification 45-46, as amended March 8, 2002.)

DISCUSSION

Claim 1 stands rejected under 35 U.S.C. § 101 "because the claimed invention is drawn to an invention with no apparent or disclosed specific and substantial credible utility or a well established utility." (Answer 3.)

The rejection of the peptide of claim 1 is predicated on the argument that the Specification does not disclose whether the fragments are up-regulated or down-regulated relative to the disease state (*id.* at 6), and thus

the Specification has not established the utility of the peptides as biomarkers for Alzheimer's disease.

As acknowledged by the Examiner, the Specification teaches that the peptide of residues 2-18 SEQ ID NO:1 is a fragment plasma protease (C1) inhibitor (*id.* at 5). Thus, if we take a step back and look at the subject matter of the claim, at bottom, claim 1 is limited to a peptide fragment of a known protein, plasma protease (C1) inhibitor. For example, Walker teaches the identification of C1 inhibitor in brain tissue by using multiple antibodies to the native protein (Walker,[2] abstract). Walker also teaches that C1 inhibitor is occasionally present in trace amounts in the senile plaques of Alzheimer's disease (Walker, p. 80, column 2).

Thus, peptide fragments derived from plasma protease (C1) inhibitor would have the well established utility as an antigen for the generation of antibodies which can be used to localize or assay for the protein. We note in addition that the Specification also discloses that antibodies may be raised to the markers disclosed by the invention (Specification 49-54). Thus, as we conclude that the peptide of residues 2-18 of SEQ ID NO:1 would have the well established utility of generating antibodies specific for plasma protease (C1) inhibitor, we are compelled to reverse the rejection.

The Examiner also rejected claim 1 under 35 U.S.C. § 112, first paragraph, on the grounds that "since the claimed invention is not supported by either a clear asserted utility or a well established utility . . . one skilled in the art clearly would not know how to use the claimed invention." (Answer 8.) This rejection is also reversed for the reasons set forth above.

---

[2] Walker et al., "Complement C1 is produced by brain tissue and is cleaved in Alzheimer disease," *Brain Research*, Vol. 675, pp. 75-82 (1995).

OTHER ISSUES

Upon return of the administrative file, the Examiner should reevaluate the patentability of the claim in view of the prior art.

As acknowledged in the Specification, residues 2-18 of SEQ ID NO:1 is a peptide fragment of plasma protease (C1) inhibitor (Specification 45-46). Moreover, the Specification teaches that specific sequences were determined as a fit to those from the theoretical digestion and fragmentation of the genome. Thus, SEQ ID NO:1 is a trypsin fragment of a known protein sequence.

In this regard we cite Carter,[3] which is drawn to amino acid sequencing of trypsin fragment of C1 inhibitor (abstract). Fragment T-34 appears to correspond exactly to residues 2-18 of SEQ ID NO:1 (page 164, Figure 1).

CONCLUSION

In summary, we reverse the rejections under 35 U.S.C. § 101 and 35 U.S.C. § 112, first paragraph, but raise other issues as to the patentability of claim 1 that the Examiner may wish to address upon receipt of the administrative file.

REVERSED

[3] Carter et al., "Genomic and cDNA cloning of the human C1 inhibitor," *Eur. J. Biochem.*, pp. 163-169 (1988), copy included.

Appeal 2007-3905
Application 09/991,799
lp

MCHALE & SLAVIN, P.A.
2855 PGA BLVD
PALM BEACH GARDENS FL 33410